REMARKS

Applicant has carefully reviewed and considered the Office Action mailed on June 10, 2008, and the references cited therewith.

Claims 3, 7, and 9-13 are now pending in this application.

'103 Rejection of the Claims

Claims 3, 10 and 11 were rejected under 35 USC § 103(a) as being unpatentable over Goggins, (U.S.Patent Application Publication number 2005/0069924A1) and further in view of Palmisano et al. (Cancer Research 60: 5954-5958, November 1, 2000 and WO 02/00927 A2. Applicant traverses the rejection.

The Examiner points to Goggins as teaching the methylation of the PAX5 gene promoter CpG islands. The Examiner further states that absent evidence to the contrary the disclosed PAX5 described in Goggins reads on PAX5 β. Applicant has reviewed the specification and teaching of the Goggins reference and found that the PAX5 sequence disclosed in Goggins fails to correspond to the PAX5 β gene promoter.

It is known that the PAX5 α and PAX5 β genes are located on chromosome 9p13 and their respective promoters are about 6.3 Kb apart. As a result, two alternative 5' exons (α and β) are spliced to common coding sequences of exons (2-10) to produce the PAX5 α and PAX5 β genes. Goggins identified in Table 1A of the Goggins reference sequence MICP8 that is 548 bp in length having homology to what Goggins identified as PAX5. The specification identified the sequence having homology for PAX5 as MICP7 (see specification at paragraph 106). A review of sequences in the sequence listing of Goggins identified SEQ ID NO. 7 having a sequence of 548 bp that might be the sequence that correlated to the gene of interest. SEQ ID NO 7 has sequence homology to a region 1161 bp upstream of the PAX5 α transcriptional start site. Therefore this sequence was well outside of the promoter region of the PAX5 α gene. Further the sequence of SEQ ID NO. 7 is about 7950 bp upstream of the transcriptional start site of PAX5 β and separated from the transcriptional start site by Exon 1 of the PAX5 α gene. Therefore since SEQ ID NO 7 of Goggins was so remote from the promoter region of PAX5 β Goggins could not be viewed as teaching the use of PAX5 β to determine the methylation state

of the promoter region of the gene as claimed by applicant since this sequence was not even in the same general proximity of this gene. Any methylation associated with the Goggins sequence is unrelated to the PAX5 β gene.

Palmisano fails to remedy the deficiency of Goggins as Palmisano fails to teach a method of monitoring for cancer in a biological specimen containing DNA from cells suspected of being cancerous and having PAX5 β gene-specific promoter methylation. Since neither reference teaches all of the elements of Applicant's claim 3, independent claim 3 is patentable over Goggins and Palmisano. Claims 10 and 11 depend from claim 3 and are patentable as least for the reasons stated in support of claim 3.

Claims 3, 10, and 11 are rejected under 35 USC § 103(a) as being unpatentable over Goggins, (U.S.Patent Application Publication number 2005/0069924A1) and further in view of Olek (WO 02/00927 A2). Applicant traverses the rejection for the reasons stated above, namely Goggins fails to teach the methylation of PAX5 β since the Goggins sequence was not even in the same general proximity of the PAX5 β gene. Any methylation associated with this sequence is unrelated to the PAX5 β gene. Olek fails to remedy the deficiency.

Therefore independent claim 3 is patentable over Goggins and Olek as neither reference teaches this element of Applicant's claim 3. Claims 10 and 11 depend from claim 3 and are patentable as least for the reasons stated in support of claim 3.

Conclusion

Applicant respectfully submits that the claims are in condition for allowance and notification to that effect is earnestly requested. The Examiner is invited to telephone Applicant's attorney (505-998-6134) to facilitate prosecution of this application.

If necessary, please charge any additional fees or credit overpayment to Deposit Account No. 13-4213

Respectfully submitted,

Janeen Vilven, Reg. No. 47,156
Direct line (505) 998-6134

PEACOCK MYERS, P.C.
Attorneys for Applicants
Post Office Box 26927
Albuquerque, New Mexico 87125-6927
Telephone: (505) 998-6134
Facsimile: (505) 243-2542
Customer No.: 005179

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